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Poorly Compressible Leg Arteries: A Specific Presentation of Peripheral Artery Disease

We commend Arain et al. (1) for their elegant study comparing the survival of patients with peripheral artery disease (PAD) and poorly compressible arteries (PCA) who were referred to their vascular laboratory. They supported previous findings, in both the general population and the clinical setting, that individuals with a very high ankle-brachial index (ABI) are at increased risk of mortality (2,3). They also suggested that patients with PCA have a poorer prognosis compared with patients with PAD. This assertion may be due to some misinterpretations and residual confounding factors.

Actually, their PAD group included patients with mild disease who had an ABI at rest >0.90 but <0.90 after 1 minute of exercise. Also, they calculated ABI by taking the lowest ankle artery pressure, so those who had only 1 diseased ankle artery were included in the PAD group, whereas to be included in the PCA group, patients should have both ankle arteries stiffened. Altogether, the authors were more sensitive to include mild PAD, but very specific to define PCA. The authors redid the analyses taking the highest pressure between ankle arteries, decreasing the hazard ratio for mortality in case of PCA versus PAD, at the limits of statistical significance. It is unclear how patients with PAD in one limb but PCA in the other limb were classified. This situation has been observed in 10% of diabetic patients who were referred to our vascular laboratory (3).

In their multivariate analysis, the authors might have missed 2 residual confounding factors: First, the models were adjusted to medications at baseline, but patients with PAD might have received more preventive treatments after the diagnosis, including statins, antiplatelet drugs, and angiotensin-converting enzyme inhibitors, compared with patients with PCA, because at that time the increased risk of the latter group was unknown. Of note, the use of angiotensin-converting enzyme inhibitors has not been included in the models. A recent analysis of the National Health and Nutrition Examination Survey cohort showed that patients with an ABI <0.90 who took at least 2 of the 3 drug groups mentioned would have an improved prognosis (4). Second, beyond adjustments for age and diabetes—2 major risk factors for PCA—the duration of diabetes per se is an independent factor associated with this condition (3,5), a factor not studied by the authors.

Finally, the authors show that the coexistence of PAD as revealed by Doppler is an independent predictor of mortality in patients with PCA, in line with our study (3). Whether patients with isolated PCA remained at increased risk of mortality compared with the PAD group has not been addressed. We found that diabetic patients with PCA without concomitant PAD had a survival equivalent to that of patients with normal ABI (3). In their series, a substantial proportion of patients with PCA had critical limb ischemia, twice as frequent as in the PAD group. We believe that the higher risk reported in patients with PCA versus PAD is related to a high proportion of concomitant severe PAD in the PCA group, compared with less severe disease in the PAD group.

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Reply

We thank Dr. Aboyans and colleagues for their interest in our study (1). They remark that our observation of a higher risk of death in patients with poorly compressible arteries (PCA) than in patients with peripheral arterial disease (PAD) may have been due to inclusion of patients with an abnormal post-exercise ankle-brachial index (ABI) and use of the lower ankle pressure to calculate the ABI. An ABI <0.9 after exercise is an established vascular laboratory criterion for PAD, and therefore such patients were included in our analyses. We used the lower of the 2 ankle pressures to calculate the ABI in each leg because such an approach identifies a greater number of patients at risk for adverse cardio-